Undiagnosed Abdominal Compartment Syndrome Causing Sudden Cardiac Arrest In Necrotizing Enterocolitis: A Case Report

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Introduction

Abdominal Compartment Syndrome (ACS) has tremendous relevance in the care of critically ill patients, because of the effects of increased pressure within the confined space of the abdomen can impair venous return and cause sudden cardiovascular collapse. ACS can encompass many diverse disease states and clinical scenarios. The problem can be acute, chronic or even undiagnosed secondary to an acute increase in intra abdominal pressure. We present a case report of a patient with necrotizing enterocolitis who had a sudden cardiovascular collapse leading to cardiac arrest from an undiagnosed abdominal compartment syndrome due to massive silent intraperitoneal bleeding.

Case

A 20 day old male, 1.4kg, with pmhx of ex 28 week prematurity, necrotizing enterocolitis, hypothyroidism, respiratory distress syndrome, patent foramen ovale, thrombocytopenia, and sepsis is brought down to the operating room for an emergent exploratory laparatomy and colon resection. Patient was brought to OR from NICU in critical conditions, with uncuffed 3.0 ETT in situ and 2 PIV in place.

A few minutes after patient was connected to the ventilator, saturation started going down from high 90’s to 80’s. Patient was taken off the ventilator and manually hand ventilated with no improvement in saturation. End tidal CO2 was not appreciated on the ventilator and at the same time, heart rate also started to go down to high 100’s. Patient was given IVP Atropine 100mcg, and the ETT was suctioned. Patient still did not have any improvement in saturation and both pulse oximetry and heart rate further kept going down.

On auscultations, very faint breath sounds were heard and still no etco2 was appreciated, so decision was made to replace the ETT under direction visualization. Again, no etco2 was appreciated on the ventilator and only faint breath sounds were heard. Pneumothorax was suspected and a needle decompression was done with no improvement in saturation. Blood pressure and saturation were unmeasurable. Chest compressions were started for heart rate below 60’s and patient was given epinephrine 20mcg twice. Epinephrine seemed to have little if any effect on patient’s vital signs.

At this point the abdomen was palpated and was found to be much distended. Abdominal compartment syndrome was suspected and the belly was immediately cut. As soon as the belly was opened, et co2 started registering on the ventilator as compression against the IVC was lifted, and
saturation and hear rate started picking up. Surgeon continued with the procedure and found severe intraperitoneal bleeding and hematoma which was evacuated and resected.

Patient was finally stabilized and resuscitated with D10 1/2NS, prbc, ffp, platelets, albumin, calcium and hydrocortisone. Dopamine drip was started. Blood loss was 100ml. Patient was still oozing during transport to NICU and DIC was suspected. Patient subsequently expired few days later from DIC and sepsis.

Conclusion

While most of the literature details the management of Abdominal Compartment Syndrome following abdominal trauma, one must keep in mind that ACS can occur in a variety of settings, particularly those associated with coagulopathy, major hemorrhage, and massive volume resuscitation such as in necrotizing enterocolitis. While ACS is usually associated with abdominal wall defects such as gastroschisis or omphalocele, one must be vigilant that silent intraperitoneal bleeding in NEC can also put these patients at risk for the development of increased intraabdominal pressure and cardiovascular collapse.